Cognitive and Perceptual Performance Effects of Controlled Exposure to Acute Hypoxic Stress

Recently, during a routine training mission, a USAF F-22 Raptor vanished from radar without adverse indications and crashed into a snow covered valley, killing the pilot. Flying conditions were ideal and no obvious cause could be discerned. The aviation mishap investigation revealed that the pilot lost consciousness due to a malfunctioning On-Board Oxygen Generating System (OBOGS). Although mishap and hazard reports suggest that OBOGS failures are isolated incidents, the majority of reported hypoxic events result from faulty O₂ delivery systems. Since FY-2000, DoD has reported four hypoxia-related Class A mishaps costing four pilots’ their lives and over $300 million in aircraft. In addition, the F/A-18 community has filed 113 hypoxia-related HAZREPS since 2001. A 2010 survey conducted on tactical aviators indicated that 79% of hypoxic episodes go unreported, suggesting that the problem is far more prevalent than official statistics suggest (Deussing et al., 2011). Current emergency procedures for hypoxic events require pilots to self-administer supplemental O₂, descend below 10,000 ft, recover, and land as soon as possible. However, the time required for individuals to fully recover cognitive and physiological functions following hypoxic exposure remains undetermined. Recent experiments indicate that performance continues to be impaired by exposure to hypoxic stress for a significant period of time after subjects are returned to sea-level equivalent air mixtures (Phillips et al., 2009).

A recent NAMRU-D experiment provided a more comprehensive understanding of the functional state of pilots and flight crew during and immediately following hypoxia exposure. Hypoxia-associated effects on specific cognitive and perceptual tasks were documented, as well as the time required for full restoration of cognitive and perceptual performance to pre-exposure levels. Twenty naval aviation candidates practiced a cognitive perceptual performance battery for 30 minutes per day for two days prior to hypoxic exposure. The performance battery included measures of visual acuity, visual contrast sensitivity, color vision, simple reaction time, choice reaction time, and executive function. After completing both practice sessions, participants returned the following day to complete the performance battery under hypoxic conditions. Subjects were exposed to a reduced-O₂ gas mixture equivalent to 18,000ft through the Reduced Oxygen Breathing Device (ROBD) for a period of 30 minutes or until their blood O₂ saturation levels dropped below 50%. Following exposure, subjects were administered a sea-level equivalent gas mixture over a 30-minute recovery period. Subjects then completed three additional 30-minute follow-up assessments while breathing sea-level equivalent air to determine when performance returned to pre-exposure levels. The follow-up assessments began at 60 minutes, 120 minutes, and 24 hours post-exposure.

The level of hypoxic stress imposed on subjects in this experiment significantly affected their performance on the simple and choice reaction time tests. Additionally, performance on simple and choice reaction time failed to consistently return to pre-exposure levels during the recovery assessments that began approximately 30, 60, and 120 minutes after hypoxia exposure (Figure 1). There were also apparent negative hypoxia-associated effects for visual contrast sensitivity. Unlike effects found for simple and choice reaction time, deficits in contrast sensitivity disappeared immediately following return to sea-level air mixtures (Figure 2). No significant effects were found to be associated with hypoxia exposure for visual acuity, color vision, or executive function.
their psychometric insensitivity to the performance effects of hypoxia due to lack of difficulty. More targeted studies are required to establish the severity and duration of hypoxia’s effect on specific cognitive and perceptual processes, as well as the time required for restoration of function following exposure. The severity and duration of hypoxia’s effect on operator functionality can best be established both through experiments documenting performance effects on validated measures of specific cognitive/perceptual abilities and through experiments designed to measure the cerebral hemodynamic response to hypoxic stimuli using modern measures such as fMRI, PET, and Transcranial Doppler.

These results provide a more comprehensive picture of the effects of hypoxia on specific cognitive and perceptual processes, and the duration of hypoxic effects post-exposure. In the case of simple and choice reaction time, effects became apparent within 10 to 20 minutes of exposure and did not subside consistently for up to 24 hours after exposure. The observed effects on visual contrast sensitivity also became apparent within the first 10 minutes of exposure, yet disappeared within 10 minutes of subjects’ return to sea-level air mixtures. No significant hypoxia-related effects were apparent in tests of executive function, visual accommodation, or color vision. This pattern of results suggests that hypoxia affects specific cognitive and perceptual abilities differently. Visual perception was affected soon after the onset of hypoxic stress, but rebounded immediately following return to sea-level air mixtures. Like visual contrast sensitivity, tests of reaction time appear to be affected soon after the onset of hypoxia, but continue to show effects beyond two hours, and potentially for up to 24 hours following return to normoxic conditions. In contrast, some cognitive and perceptual abilities, such as executive function, visual accommodation, and color vision do not appear to be affected by the level of hypoxic exposure imposed during this experiment.

The lingering effects of hypoxia on simple and choice reaction time supports the conclusion by Phillips et al. (2009), that some aspects of performance do not recover immediately after subjects are returned to a sea-level equivalent environment. In the case of simple and choice reaction time, consistent return to baseline performance was not observed during the 30, 60, or 120-minute recovery assessments (Figure 1). The lack of hypoxia-associated effects on executive function, color vision, and visual accommodation may suggest that these processes remain intact until moments before a subject loses consciousness. With regard to executive function, this interpretation is consistent with the hypothesis that hypoxia-related deficits observed in laboratory settings are the result of a general perceptual slowing and do not necessarily imply that disruptions in higher-order cognition have occurred (Lindeis, Nathoo, & Fowler, 1996). However, the lack of performance effects on tests of executive function, color vision, and visual acuity may have resulted from their psychometric insensitivity to the performance effects of hypoxia due to lack of difficulty. More targeted studies are required to establish the severity and duration of hypoxia’s effect on specific cognitive and perceptual processes, as well as the time required for restoration of function following exposure. The severity and duration of hypoxia’s effect on operator functionality can best be established both through experiments documenting performance effects on validated measures of specific cognitive/perceptual abilities and through experiments designed to measure the cerebral hemodynamic response to hypoxic stimuli using modern measures such as fMRI, PET, and Transcranial Doppler.

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